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Ontario
Ministry of
Labour

Occupational
Health and Safety
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*Occupational
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Occupational Health in Ontario

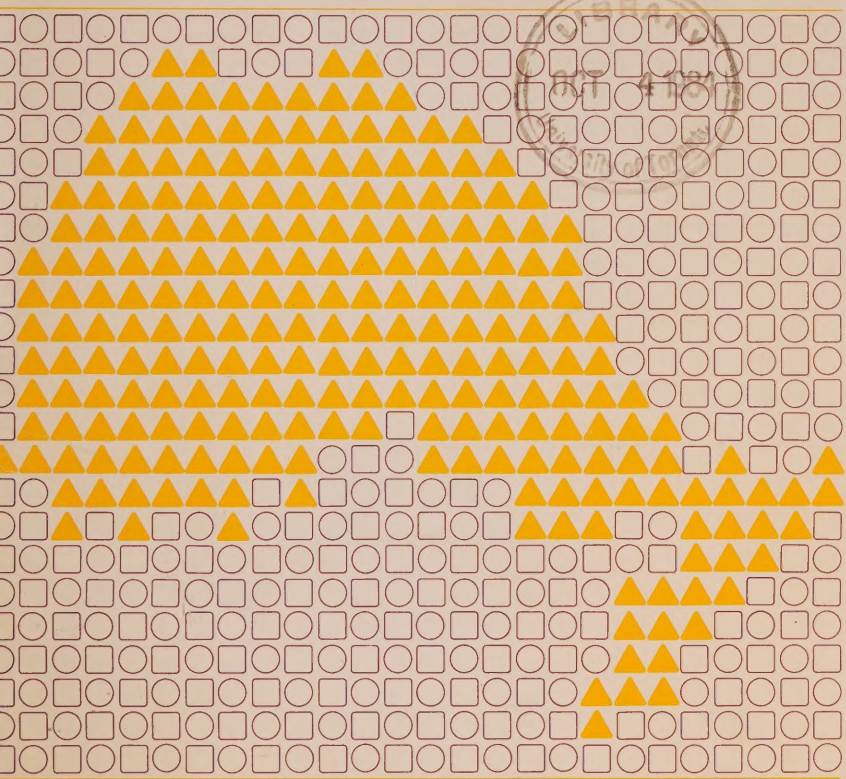
Volume 2, Number 1 January, 1981

Hazards in Histo-Technology

Nitroglycerin and Medical Surveillance

Nepheline Syenite Pneumoconiosis

Outpatient Treatment of Burns in Industry



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Hazards in Histo-Technology*

Summary

This paper reviews the hazards in histo-technology laboratories. The physical, biological and chemical hazards more frequently encountered are enumerated and briefly described. The necessary precautionary measures are outlined.

One can only reflect with amazement on the risks that laboratory workers used to take before the dangers of x-rays, radium, benzene, carbon tetrachloride, mercury, asbestos, etc., were made known. Indeed, most senior laboratory technicians can still relate astounding anecdotes about various hazards. In spite of the potential dangers inherent in the laboratory situation, some workers may feel, even today, that it is not worth taking precautions to avoid a remote risk of injury or accident. However, an attempt should always be made to preclude an untoward result from happening, as such action may save a life or prevent serious injury.

Safety must be accepted as an integral part of everyday thinking and working, and not be regarded as an additional burden.[1] Only with this philosophy will one be able to assess successfully one's working facilities.

Because of the variety of materials utilized in a laboratory, it is important that a systematic approach to hazard identification be adopted. Furthermore, safety aspects need to be considered in advance when designing an experiment or planning a new procedure, always appreciating that even the simplest apparatus can present a potential danger.

Hazards can be most logically classified into nine categories[2]: mechanical, electrical, ionizing, non-ionizing, microbiological, chemical, atmospheric pressure differentials, thermal non-fire, and fire.

Some of these hazards, such as ionizing and non-ionizing radiation, which would be present in a nuclear medicine facility, are not likely to be found in an histology laboratory; while microbiological hazards, obvious in a microbiology department, may exist in all departments where specimens are handled, including the histology laboratory.

The hazards encountered most frequently during the workday in the histology laboratory are microbiological, physical, fire and chemical.

*Given at the Ontario Society of Medical Technologists Meeting, Toronto, September 25, 1980.

Biohazards

The risk of bacterial, mycotic and viral infections from fresh (unfixed) or partially fixed tissues removed at surgery or autopsy can be considerable. The degree of risk generally depends upon the worker's resistance, the portal of entry, the exposure time, the virulence of the micro-organism or potency of the toxin, and the total dose received. Micro-organisms on the skin can gain entry into the body via cuts and abrasions or through mucous membranes. Inhalation exposure is possible when micro-organisms are accidentally released in a fine spray, for example during a spill. Ingestion occurs when micro-organisms are transferred to the mouth by fingers, cigarette or pipe or during consumption of food or drink.

To minimize potential dangers to laboratory personnel, all specimens should be considered infectious and handled accordingly.

It is good practice to separate the room used for receiving and cutting specimens from the rest of the laboratory, and to exercise the same precautions as adopted in the microbiology department. Rubber gloves should always be worn and, after each cutting session, all instruments and cutting boards should be thoroughly cleaned and disinfected. The most important factor in preventing the spread of infection is proper handwashing.

Micro-organisms can be divided, according to their pathogenicity, into two categories as shown in Table 1.[3] Category A pathogens are very rare virulent viruses and are almost never encountered in the hospital laboratory. Category B pathogens include the more commonly encountered organisms; the one probably most frequently found in the histology laboratory up to now is mycobacterium tuberculosis.

Because of potential infection, all biological waste should be double-bagged using identifiable coloured garbage bags, and frozen for transportation. The selected way of disposal is by incineration.

Physical Hazards

A high level of personal hygiene and good housekeeping practice is essential for laboratory safety, and all personnel should ensure that their work habits do not endanger others working nearby. Many minor accidents resulting from poor laboratory practice can be avoided with a little forethought and by remembering to work tidily. It is always important to clear used apparatus and materials, wash up promptly, attend to spills immediately, and maintain clear pathways and

corridors. Laboratories should be as clean and orderly as possible with all items securely and safely stored.

The following protective equipment should be readily available and appropriately placed: safety goggles and face shields, eye-wash fountains or eye-wash (squeeze) bottles, fire extinguishers, fire blankets and an emergency water shower. All staff should be instructed in the proper use and location of such equipment.

No eating, drinking or smoking should be permitted in the histology laboratory, and mouth suction to fill pipettes should never be used.

Although the importance of body mechanics has been stressed many times with respect to work and leisure activities, improper posture during lifting still results in innumerable injuries. It is important to use leg muscles when lifting, and it should be remembered never to lift and twist at the same time, nor to lift heavy objects high and away from the body.

Care should be exercised when handling, cleaning and washing glassware. Glass tubing and ampoules should be cut with a glass-knife made of tungsten carbide rather than with a file.

Section-cutting knives, scalpels, and needles for injection are all potentially dangerous. No attempt should be made to arrest the fall of any of these from hands, bench or microtome, as a serious wound could result. The microtome should always be put in the safe (locked) position when manipulating the wax blocks. Freezing microtomes in quick section rooms are a source of additional danger because of low temperature and ice formation which may cause burns.

Burns are also possible from electrical or thermal equipment and bunsen burners.

Electrical equipment always presents a potential hazard in the histology laboratory because of the multiplicity of equipment and the use of high electrical power. Good, planned maintenance, in addition to supplier contract servicing, is essential. Corrosion of electrical leads by chemicals is an added hazard in the laboratory, requiring prompt attention, and electrical units which are to be operated in an area exposed to flammable vapours should be explosion-proof. All electrical equipment should be earthed and Canadian Standards Association approved.

Ventilation in the laboratory is a major concern because of toxic inhalation risks and, depending on the specific purpose, both general and local exhaust ventilation may be used. Local exhaust ventilation should be provided for tissue processors, mounting

procedures and specimen storage areas. Obviously, the exhaust should be directed to the outside to avoid contamination of the rest of the hospital. This stack emission may require approval from the Ministry of the Environment.

Cylinders with compressed gases are obviously dangerous and should be handled very carefully. They must always be securely fastened whether in storage, transit or use, and valves and controls should be treated with respect.

By adopting these general safety rules, serious accidents and deaths should be minimized, if not completely prevented.

Finally – if in doubt, one should always ask.

Many accidents are caused by failure to understand equipment, and failure to seek advice or information. If laboratory method cards or books are used, they should contain safety details at the appropriate place in the text.

Fire Hazards

Fire is the principal cause of serious laboratory accidents and usually involves organic solvents. Nearly all organic solvents are flammable, some extremely so because of their high vapour pressure at room temperature. Since most organic vapours or gases are heavier than air, they tend to flow along surfaces and settle on the floor. Therefore, open flames, or other heat sources quite distant from the source of vapours, can cause a flashback and fire or explosion.

In discussing flammability, one needs to be reminded of some definitions.

The *flash point* of a liquid is the temperature at which it gives off sufficient vapour to form an ignitable mixture with the air near the surface of the liquid or within the vessel used (Table 2).

If the flash point of a liquid is below room temperature (25°C) it is termed a Class I liquid and is most dangerous[4] (Table 3).

The *auto-ignition* temperature of the liquid is the minimum temperature required to initiate or cause self-sustained combustion independent of applied heat.

The *evaporation rate* is the ratio of the time required to evaporate a measured volume of one solvent to the time required to evaporate the same volume of a reference solvent (ethyl ether) under identical test conditions. This is important in considering solvent mixtures with different evaporation rates.

Explosive range or flammability limits are expressed in per cent by volume of fuel vapour in air, and are

ranges of concentration over which a particular vapour or gas mixture will burn when ignited. Explosions usually result from extremely rapid exothermic reactions (usually oxidation) of mixtures of compounds. As heat is evolved, the reaction rate increases and often large volumes of expanding gases are produced in fractions of a second.

Fires are usually classified according to the type of material being consumed. It is important to use the proper type of extinguisher for each class of fire, so as to control the situation quickly and avoid compounding the problem as illustrated in Table 4.[4]

Flammable liquids are best kept in well-ventilated storage rooms or at floor level away from sources of heat in specially constructed cabinets. They should never be stored with oxidizing agents. Only approved containers should be used and the size of these is determined by the class of fluid according to U.S. National Fire Protection Association.[5] Great care should be exercised to avoid possible ignition sources such as smoking, electric equipment, open flames, static electricity, oxidizing agents, etc., in the areas where flammable liquids are stored or used.

Transfer of solvent from one metal container to another can produce a voltage potential that can result in a static spark capable of ignition; therefore if both are metal, two containers must be bonded before pouring.

Flammable liquids should not be disposed of down the drain.

In histology laboratories, large quantities of flammable material are used, e.g. solvents, paraffin wax, celloidin; so extra care is required.

The paraffin oven bases must be kept clean and free from wax, and melted wax must be dispensed carefully when "blocking" tissues.

Because of the flammable nature of reagents used and the toxic fumes produced, tissue processors should be in a separate room equipped with a sprinkler or CO_2 system, an exhaust fan and a fire door.

Special precautions against fire hazards are essential in laboratories where celloidin or nitrocellulose is used, e.g. in neuropathology.

Hazardous Chemicals

Chemicals represent perhaps the greatest number and variety of dangers in the histology lab. One of their major hazards is fire, which has already been discussed. The other potential perils include toxicity,

carcinogenic and teratogenic effects. These risks are further complicated by complex reaction combinations.

Toxic compounds enter the body through the lungs, the gastrointestinal tract and the skin, as shown in Figure 1.

They may produce effects at the site of contamination, be absorbed into the circulation and produce effects on specific target organs, or interfere with basic life-supporting reactions at many sites in the body.

Local injury of the skin by solvents, cleansers and disinfectants is very common. Chemicals may act as primary irritants, as allergic sensitizers or as photosensitizers. A primary skin irritant is a substance that causes damage at the site of contact because of its direct chemical or physical action on the skin, for example, its pH. No antecedent immunologic sensitization is required. However, a primary skin irritant may also act as an allergic sensitizer, for example, formaldehyde. Only about 20 per cent of all occupational dermatitis stems from skin allergies, the remaining 80 per cent being a reaction to primary irritants.

The chemicals causing allergic dermatitis are fewer in number than those causing primary irritation but the reactions may be more severe. Prevention of dermatitis can be accomplished by environmental controls (material selection, good housekeeping), personal protective measures (cleanliness, gloves, barrier agents) and by administrative procedures (pre-placement examinations, health education, job rotation, periodic monitoring).

Some chemicals may be absorbed across the skin and produce systemic effects. The rate of absorption depends on numerous factors including molecular weight, lipid solubility, corrosive properties and the integrity of the skin. Mucous membranes, including eyes, are even more susceptible to irritation, and absorption occurs in these areas more readily than through the skin.

The respiratory tract is the commonest route of absorption and the depth to which materials penetrate depends on their physical properties. Small particles, up to five microns in diameter, usually reach the alveoli, whereas aerosols in excess of 30 microns may not be able to penetrate beyond the nose. In the case of true gases and vapours, the site of action mainly reflects differences in solubility. Highly soluble materials (ammonia, hydrogen chloride and formaldehyde) affect primarily the upper respiratory tract. Compounds of intermediate solubility (halogens, ozone,

diethyl or dimethyl sulfate and phosphorous chloride) affect both the upper respiratory tract and pulmonary tissue. Insoluble materials such as nitrogen dioxide, arsenic trichloride and phosgene affect primarily the lungs. Irritation of the upper respiratory tract leads to rhinitis, pharyngitis and laryngitis; irritation of the bronchi leads to tracheobronchitis, bronchitis and pneumonia; irritation of the lungs leads to pulmonary edema and pneumonia.

Ingestion is not a common route of absorption in the laboratory, but it is possible. The dust collected in the mucus of the upper respiratory tract may be swallowed, or food and drink may become contaminated.

In the body, toxins can accumulate at the various target sites (protein, fat, bone), or can be metabolized to non-toxic end products (detoxification) or to a more toxic product, e.g. methanol → formaldehyde (toxic) → formic acid (toxic).

The liver is the organ most active in the biotransformation of toxins, but enzymes in other tissues such as esterases in the plasma and enzymes in the kidneys, lungs and gastrointestinal tract may also metabolize chemicals. Excretion is via expired air, the alimentary tract either directly or in the bile after absorption into the hepatic circulation, the urine, saliva and sweat.

The presence of metabolites in urine e.g. hippuric acid from toluene, phenol from benzene, toluic acid from xylene, and trichloroacetic acid from trichlorethylene is indicative of exposure, but not necessarily of poisoning. It should be noted that some of these tests are of limited value because, for example, hippuric acid, originating from a diet containing benzoic acid or its precursors, is a normal constituent of the urine.

Various compounds can act synergistically. By blocking a metabolic pathway, one toxin may reduce the effective metabolism of another, and therefore enhance its toxic effects. For example, if a mixture of carbon tetrachloride and benzene is absorbed, the former will affect the liver and may interfere with benzene conjugation.

Safety standards have been improved by the use of Threshold Limit Values (TLV). A short-term exposure limit (STEL) or a time-weighted-average (TWA) for an eight-hour workday and 40-hour work week are used. Airborne concentrations of substances are expressed as ppm or mg/m^3 in the volume of air. A TLV list is published annually by the American Conference of Governmental Industrial Hygienists.[6] The TLVs repre-

sent levels at or below which it is unlikely that impairment of health will occur in a normal person. They must not be considered as upper limits of safety, however, because some susceptible individuals may be affected below these levels, and skin absorption may occur.

In an histology laboratory, the hazard from toxic vapour production is increased because of the large quantities of organic solvents present. The nature and extent of the hazards related to their use is dependent upon the physical, chemical and toxicologic properties of the individual solvent. Harmful effects come principally from flammability, skin contact and inhalation of vapour. Most laboratory solvents will dissolve the protecting fats and oils in the skin and cause dermatitis. Precautionary measures should include the use of protective clothing, suitable barrier creams, eye and face protection, and good personal hygiene. Local and general ventilation must be adequate, and respiratory equipment available. Fire protection for Class B fires is appropriate.

Formaldehyde[7] is a colourless gas with an irritating odour. It is soluble in water, alcohol and ether. Commercially, it is available in aqueous solution and stabilized grades contain methanol, because it polymerises readily in both the liquid and solid state. In the histology laboratory it is used extensively in various concentrations of formalin (40 per cent formaldehyde by weight in water) for the purpose of fixing and storing specimens.

Formaldehyde is absorbed rapidly by all routes of entry and disappears rapidly from the blood. It is oxidized to formic acid, and partly excreted as formate in the urine. The formic acid may be further oxidized to CO_2 and water.

Exposure to low atmospheric concentrations of vapour causes irritation of the eyes and respiratory tract. Because of its solubility, the effect of formaldehyde is most noticeable in the upper regions of the respiratory tract. The odour threshold is at or below 1 ppm, and the ability to perceive the odour is blunted within one or two hours of exposure, but it returns when the exposure is interrupted. A concentration of 1-2 ppm may be irritating or annoying to some individuals, but most people will only experience slight discomfort at 2-3 ppm. Discomfort increases with concentration; 10 ppm is tolerated with difficulty even briefly, and between 10 and 20 ppm there is intense lacrimation, burning of nose and trachea with difficult

breathing and cough. Exposure to 50 to 100 ppm produces headache, feeling of restricted chest, pulmonary edema and, in extreme cases, death, caused by edema or spasm of the glottis. Asthmatic symptoms from allergic sensitivity may occur even at low concentrations. The current TLV is 2 ppm.

Dermatitis is the most common occupational injury from formaldehyde since it is both a primary irritant and a potent sensitizer. Cases of urticaria and nail lesions have been also reported. Primary irritation of the skin has been caused by solutions as dilute as 4 per cent formaldehyde (10 per cent formalin), and sensitized individuals can react to 1:5,000,000 - 1:8,000,000 dilutions as well as to the vapour in concentrations of 10 ppm.

It is generally recognized that individuals once sensitized to formaldehyde should avoid further exposure to it in any form.

The area where formaldehyde is used should be well-ventilated (may require local exhaust ventilation with fumehoods) and, if the TLV is exceeded, proper respiratory equipment should be used. Formaldehyde and its vapours are flammable and the usual precautions should be taken.

Protective clothing and equipment should be used when working with formaldehyde. Contaminated skin should be washed with soap and water, and eye splashes should be treated by irrigation for 15 minutes.

Some concern has been expressed about possible carcinogenicity, mutagenicity and teratogenicity of formaldehyde based on laboratory rat studies. Formaldehyde can react with hydrogen chloride and inorganic chlorides to yield a carcinogen, bis-chloromethyl ether (bis-CME), but this reaction occurs only at very high concentrations of precursors and there is no evidence of bis-CME being formed in occupational environments.

Xylene[8], also called dimethylbenzene or xylol, exists in three isomeric forms: ortho, metha and para. It is insoluble in water but soluble in ethyl and isopropyl alcohol, ether and other organic solvents. In the laboratory it is used extensively as a clearing agent.

Like most organic solvents it is a skin-defatting agent causing erythema and dryness and, with prolonged contact, blistering. Absorption through the skin is possible.

The vapour is also an irritant to mucous membranes including the conjunctiva and respiratory tract at

about 200 ppm. Odour threshold shows large individual variability (0.14-20 ppm), and although olfactory fatigue can develop, full recovery occurs within 10 minutes. Threshold limit value is 100 ppm.

Xylene also has narcotic effects on the central nervous system at higher concentrations, which are however rarely tolerated because of its irritating properties. Levels of 270-350 ppm cause giddiness, anorexia, headaches, vertigo, etc.

At very high concentrations it can cause transient renal and hepatic dysfunction, loss of consciousness, retrograde amnesia and death.

For many years, myelotoxicity (toxicity to the blood-forming organs) has been attributed to xylene, primarily because of the close structural similarity between xylene and benzene which is a proven myelotoxin. Xylene has been frequently contaminated with benzene in the past. Current scientific evidence obtained from human and animal studies indicates that alkylation of the benzene ring results in a loss of these bone marrow effects.

Toluene, like xylene, is primarily a narcotic. The symptoms of fatigue, weakness and confusion have been reported at exposures of 200 to 300 ppm, with nausea and loss of coordination occuring at about 600 ppm. It is a stronger irritant of the skin and mucous membranes and has therefore been largely replaced by xylene. Skin absorption does occur, but this is not a major route of entry in most work situations. There is no definite evidence to link either toluene or xylene exposure to permanent hematalogic damage. The present TLV for toluene is 100 ppm.

The toxic effects of the *alcohols*[9] from occupational use are not severe in terms of injury to the internal organs, with the exception of methanol which can cause permanent damage to ocular tissue, though this is much more rarely observed following inhalation than by ingestion. Physiologically, all alcohols are weaker narcotics than the hydrocarbons, and their narcotic powers increase with increasing molecular weight. Specific differences in the degree and nature of their toxicity are related to their metabolism. Ethanol is rapidly converted to CO_2 and H_2O, while methanol is eliminated slowly with the formation of formaldehyde and formic acid which are potent toxins to nervous tissue.

Methyl, ethyl and isopropyl alcohol are not very toxic to the skin, but they are flammable and potentially explosive.

The signs and symptoms of acute intoxication following ingestion of *methanol*[10] is similar to that from alcoholic beverages but development of blindness is a real danger. The exact reason for the selective attack on the retinal cells and ocular nerve is not known, but it appears to be related to the intermediate metabolic products of formaldehyde and formic acid.

Repeated exposure to methanol vapours may cause irritation of the mucous membranes, headaches, tinnitus, tremor, and neuritis. Blindness can also occur following exposure to its vapours, but only at very high concentrations and the hazard from this method of entry is more potential than actual. TLV is 200 ppm.

Toxic manifestations can be attenuated by the administration of ethanol, which is capable of inhibiting the metabolism of methanol and therefore permits its excretion unchanged.

Ethanol, whether ingested or inhaled, exerts its principal action on the brain, first inhibiting its higher functions and then acting as an anesthetic with a higher potential toxicity than methanol.

The amount ingested which can cause death from respiratory depression has been stated to be equivalent to approximately one quart of whiskey within a short interval.

Vapours can cause mild irritation of mucous membranes, and inhalation exposure can have effects similar to those from ingestion of the liquid. It is not clear if the effects on the liver, such as that known to arise from repeated excessive ingestion, are also caused by repeated inhalation.

The main hazard from occupational exposure is the possibility that employees may be tempted to drink it. Not only may they become habituated to it and therefore more susceptible to accidents, but it may increase their susceptibility to other poisons because of liver damage. TLV is 1,000 ppm.

While the ingestion of *isopropanol*[11] has a similar action on the central nervous system causing drowsiness, headache, narcosis and, in severe cases, coma, repeated administration has no injurious action on the liver or on the vision. It is more slowly metabolized than ethanol which may account for the fact that it is more narcotic and acutely toxic. Although the complete metabolic pathway is unknown, acetone has been identified as a metabolite.

No cases of occupational poisoning by pure isopropanol have been found in the literature, although there

are many cases known in chronic alcoholics. Odour threshold is 40-200 ppm, and concentrations from 400-800 ppm cause irritation of eyes, throat and nose. TLV is 400 ppm.

The principal safety hazards of isopropanol are fire and explosion.

Handling and Disposal of Chemicals

It is useful to have rules for the labelling, storage and handling of chemicals, because they will serve as a reminder of the potential hazard of all such materials.

Labelling is extremely important. All chemicals should be labelled as to the chemical content, danger and the date of preparation. One of the most widely used labelling criteria is provided by the National Fire Protection Association[12] as shown in Figure 2.

The system identifies three hazards: Health, Flammability, and Reactivity. The severity of each is noted by numbers from 0 to 4, with 0 indicating minimal and 4 indicating the greatest degree of hazard. A fourth area on the hazards sign is provided for special information, which might be vital information to a fire fighter. For example, W is used to indicate the presence of a material which reacts violently with water.

For safe storage under appropriate conditions without danger of incompatibility, it is necessary to evaluate the potential hazard from the physical and chemical properties of substances and classify them into groups[13]: flammable materials, oxidizing agents, water-sensitive materials, toxic chemicals and compressed gases. Some of the incompatible substances are listed in Table 5.

Flammable and combustible liquids requiring refrigeration must be stored in approved explosion-proof refrigerators because of the possibility of explosive vapour-air mixture creation.

Old samples should be discarded periodically, using proper caution.

Chemicals which can form peroxides such as ethyl ether, dioxane or tetrahydrofuran are of particular concern because they can explode if they are allowed to age and are subsequently disturbed by opening or shaking. Another example is picric acid which must be stored in water; it becomes extremely explosive if allowed to dry out.

Ammoniacal silver solutions used in silver impregnation methods tend to precipitate sensitive explosive crystals which are also liable to detonate when the container is handled; therefore solutions should be

freshly prepared before use, protected from sunlight and the remainder disposed of immediately after use.

A number of chemicals are strong corrosives (hydrochloric acid, sulfuric acid, sodium hydroxide, osmium tetroxide, potassium dichromate, chromic acid) and have to be handled appropriately.

Fixatives are poisonous chemical solutions that coagulate protein and prevent natural liquefaction. "Living" skin may also be fixed if in contact with fixatives for 15 minutes or more; therefore, if any body contact occurs the hands should be washed thoroughly and eyes must be irrigated for 15 minutes. Formol-corrosive and Zenker's Fluid both contain mercuric chloride which is extremely corrosive to metal, including lead pipes. It is also both a primary skin irritant and a potent sensitizer. Bouin's Fluid and Brasil Fixative contain picric acid. Formaldehyde is the major component of Formol-saline and 10 per cent formalin, whereas Carnoy's Fluid contains alcohol, acetic acid and chloroform. All of them should be handled with care because of possible irritant effects on mucous membranes and skin.

Formalin jars and xylol dishes should be kept covered when not in use. It is also advisable to keep the staining dishes in a fume chamber to avoid unnecessary fume buildup. All cover-slipping should also be done in the fume chamber or under exhaust ventilation.

Significant attention has recently been focused on chemical agents which are carcinogenic, e.g. azodyes. Fortunately, none of these chemicals are used in the histology lab routinely or in appreciable quantities.

Chemical spills are usually caused by breakage of bottles or flasks, and the material may be corrosive to skin, clothing or flooring and/or may produce flammable or irritating vapours. Therefore any spill should be promptly handled by isolation with absorbent material, neutralization if necessary (sodium carbonate for acid spills, boric acid for alkali spills) and then appropriately disposed of. In the case of flammable liquid spills, all sources of ignition should be shut off. The area of the spill with high vapour concentrations should be well ventilated. Respiratory protection might have to be used during the cleanup, and chemical cartridge respirators or self-contained breathing apparatus should be used as indicated.

Disposal of the large amounts of chemical waste from the laboratory is a difficult problem. Waste should be collected in safety cans and contents should be clearly indicated on the containers. Separation instructions should be based on the chemical reactivity of individual compounds. All solvents should be stored in a safety cabinet and contents burned in a solvent burner. Other wastes can be disposed of in the sewer system but have to be diluted, neutralized or pretreated to meet the Sewer Use By-law of the appropriate municipality. For example: acids and alkalis are preferably neutralized or diluted at least 50:1 before disposal; while mercury, azide, picric acid, arsenic, cyanide, chromates and phenolic compounds should be pretreated before being poured into the sewer system.

It is preferable to have all chemical wastes collected by a commercial disposal firm.

Conclusion

The Occupational Health and Safety Act (1978) in Ontario specifically refers to toxic substances used in the workplace and puts obligations on both employers and workers to identify hazards and keep their workplace safe, stressing internal responsibility. Everyone therefore needs to be concerned and participate in hazard detection and prevention. Proposed regulations have been gazetted for some designated substances specifying engineering controls, work practices, administrative controls and medical surveillance. Others will follow. Meanwhile, the Ministry of Labour's inspectorate (the Industrial Health and Safety Branch) will audit your workplace and the Occupational Health Branch staff – medical and hygiene – will support them as their consultants. Our expertise in this field will grow with experience, and we hope that jointly with you we may tackle the areas of concern in the most appropriate manner, and rectify any faults for the common good. With mutual trust and co-operation we cannot fail to succeed.

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Figure 1
Routes of Absorption, Distribution and Excretion

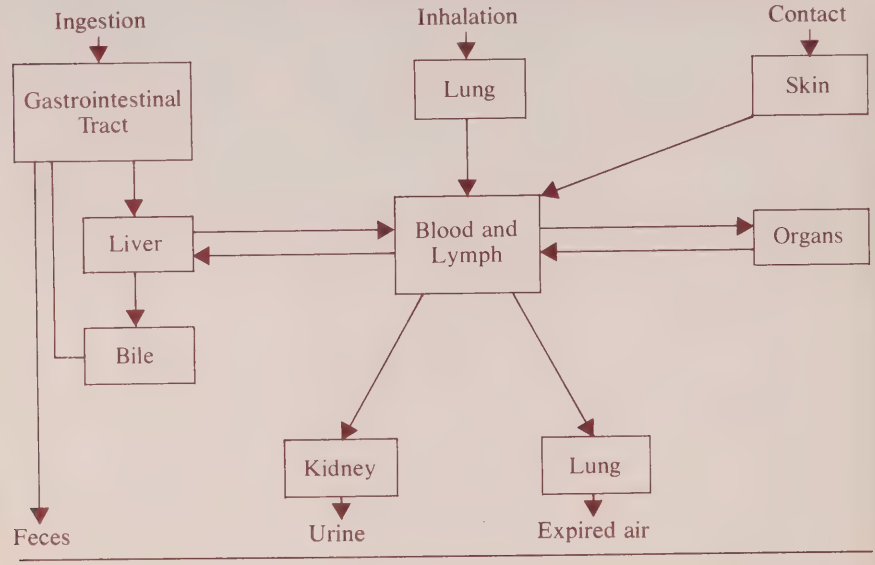

Table 1

Category A pathogens	
	Lassa-fever virus
	Marburg virus
	Rabies virus
	Simian herpes-B virus
	Smallpox virus
	Crimean-hemorrhagic-fever virus (Congo)
	Machupo-hemorrhagic-fever virus
	Junin-hemorrhagic-fever virus
	Venezuelan-equine-encephalitis virus
Category B pathogens	
Viruses	Arboviruses, all species not in Category A
	Lymphocytic-choriomeningitis virus
	Serum hepatitis (HBAg)
	Unattenuated yellow-fever virus

Bacteria	Bacillus anthracis Brucella, all species Clostridium botulinum Leptospira, all species Mycobacterium leprae Mycobacterium tuberculosis, all strains Pseudomonas mallai (Lofflerella mallei) Pseudomonas pseudomallai Salmonella typhi Vibrio cholerae Yersinia pestis (Pasteurella pestis)
Rickettsiae	Rickettsia, all species Coxiella burnettii (Q fever)
Chlamydia	Chlamydia psittaci

Figure 2 Hazard identification symbol.

Table 2
Flammables Commonly Found in Histology Laboratories

Substance	Flash Point °F
Acetic acid	109
Acetone	0
Ethyl Alcohol	54
Ethyl Ether	−49
Isopropyl Alcohol	53
Methanol	52
Toluene	40
Xylene	81

Table 3
Classification of Flammable Liquids

		Flash point	*Boiling point*
Flammable or Class I Liquid		<100°F (37.8°C)	
	IA	< 73°F (22.8°C)	<100°F (37.8°C)
	IB	< 73°F (22.8°C)	≥100°F (37.8°C)
	IC	73°F (22.8°C) ≤ fp < 100°F (37.8°C)	
Combustible Liquid		≥100°F (37.8°C)	
	II	100°F (37.8°C) ≤ fp <140°F (60°C)	
	IIIA	140°F (60°C) < fp <200°F (93.4°C)	
	IIIB	≥200°F (93.4°C)	

Table 4
Fire Extinguishers

Class of Fire	Typical Fuel Involved	Suitable Extinguisher
ORDINARY COMBUSTIBLES	Wood	Water types
	Paper	
	Cloth	Foam (very small fires only)
	Rubber	
	Plastics	
	Rubbish	Multipurpose Dry Chemical
	Upholstery	
FLAMMABLE LIQUIDS	Gasoline	Foam
	Oil	Carbon dioxide
	Grease	Halogenated agent
	Paint	Regular or ordinary dry chemical
	Lighter fluid	Multipurpose Dry Chemical
ELECTRICAL EQUIPMENT	Motors	Carbon dioxide
	Appliances	Halogenated agent
	Wiring	Regular or ordinary dry chemical
	Fuse boxes	
	Switchboards	Multipurpose Dry Chemical
COMBUSTIBLE METALS	Aluminum	Dry powder extinguishers and dry power agents only
	Magnesium	
	Potassium	
	Sodium	
	Titanium	
	Zirconium	

Table 5
Some Incompatible Laboratory Chemicals
(The omission of certain reagents does not signify lack of hazard)

Chemical	*Avoid Storage with These Substances*
Acetic acid (glacial)	Chromic and nitric acid, hydroxyl-containing compounds, ethylene glycol, perchloric acid, peroxides and permanganates. Avoid storage near combustible materials.
Acetone	Hydrogen peroxide and concentrated mixtures of nitric or sulfuric acid.
Alkalis and alkaline earth metals (sodium, potassium, cesium, lithium, magnesium, calcium)	Carbon dioxide, chlorinated hydrocarbons, water, acetaldehyde.
Ammonia (anhydrous)	Oxidizing gases (particularly chlorine, bromine, iodine) and acids. Mercury, calcium hypochlorite and hydrogen fluoride, acetaldehyde.
Hydrocarbons (benzene, butane, propane, gasoline, turpentine, etc.)	Fluorine, chlorine, bromine, chromic acid, sodium peroxide.
Hydrochloric (muriatic) acid	Chlorates, oxidizable substances, metal powders.
Ntric acid (concentrated)	Acetic acid, aniline, carbides, chlorates, chromic acid, hydrogen cyanide, hydrogen sulfide, organic acids, acetone, hydrogen bromide.
Perchloric acid	Acetic anhydride, alcohols, dehydrating agents, combustible materials, oxidizing and reducing agents.
Silver	Acetylene, ammonia compounds, oxalic acid, tartaric acid, fulminic acid, picric acid.
Sodium hydroxide	Water, nitro-paraffins, phosphorus.
Sulfuric acid	Carbides, chlorates, fluorides, perchlorates, permanganates, picrates, metallic powders, acetone.
Water	Alkali metals and oxides, alkali metal hydrides, sulfuric acid, sulfur trioxide.

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Nepheline Syenite Pneumoconiosis

Summary

Fourteen cases of nepheline syenite pneumoconiosis are reviewed. The signs and symptoms, x-ray changes, pulmonary function test results and pathology are described. Possible mechanisms of injury are discussed.

Nepheline Syenite Pneumoconiosis

Nepheline syenite pneumoconiosis is a condition mentioned in several textbooks[1,2], but few cases have been reported in the literature. The major source is an article by Barrie and Gosselin[3] in 1960 which describes the pathological findings of one of the early cases. Nepheline syenite is commercially mined in very few locations, but the Peterborough area mines in Ontario are the major producers in the western world, so the Ontario experience with nepheline syenite is probably the most extensive. This paper reviews the cases identified from this source and summarizes the current situation with regard to this particular condition.

Nepheline Syenite

Nepheline syenite is an igneous rock containing nepheline, feldspar and a variety of other minerals. It consists mainly of sodium or potassium aluminum silicates and is classed as a non-fibrous silicate. A typical analysis of grade "A" nepheline syenite reveals 60.56% SiO_2, 23.68% Al_2O_3, 9.78% Na_2O, 4.37% K_2O and smaller amounts of Fe_2O_3, CaO and MgO.[4] It characteristically contains no free silica. It is used in the glass and ceramic industries and as a filler in paints, papers, plastics and foam rubber.[5]

Canada was the sole western supplier of nepheline syenite until 1961 when mining and production were started in Norway. It has also been mined in the USSR since the 1930s, but for its phosphate and aluminum content primarily.

In Canada, the production is located near Peterborough, Ontario. Operations commenced in the winter of 1935-1936. The mining is open pit, and rock is blasted from the face. It is then transported to the mill where it is crushed, dried, screened, air classified and the iron-containing materials removed by magnetic separation. The old mill was in operation until 1956, at which time a new mill was constructed. The first cases of lung disease attributed to nepheline syenite exposure appeared in the late 1940s.

Method of Search

The records of the Advisory Committee on Occupational Chest Diseases of the Ontario Workmen's Compensation Board were searched and 16 claims were found.

These probably represent all or the majority of the claims submitted to the Ontario Workmen's Compensation Board, as the common practice is to have all such claims assessed by the advisory committee. Of the 16 persons who submitted a claim, 14 were awarded compensation. One was not thought to have significant disease. The other was a claim following death due to bronchogenic carcinoma. As there was no known association between nepheline syenite and cancer, the claim was denied.

Of the remaining 14 cases, detailed histories are available on 13, pulmonary function test results on 11, and serial chest x-rays on all. Pathological data are available on five of the claims. This forms the basis on which the observations and results are made. All claims are of persons who worked in the old mill. None of the workers gave a history of significant exposure to other known pneumoconiosis-producing dusts.

Dust Levels

No nepheline dust level measurements are available for the time period when the men were exposed to the old mill. Recent levels are known but are taken from the new mill. Workers described the conditions in the old mill as very dusty. An occupational chest disease consultant who visited the old mill reports that it was so dusty, it was almost impossible to see and recognize a person a short distance away.[6]

Results

The data, in respect to each case, are presented in Table 1. The figures for length of exposure are derived from both the Workmen's Compensation Board claim records which the company supplied, and from the history taken by the examining doctor. All but one had good agreement concerning the start of work. All 14 persons had a minimum exposure of four years in the old mill, and the exposure ranged from four to 20 years. Many were also employed in other areas of the workplace, for example, the quarry. The length of time between initial exposure in the old mill and the onset of symptoms ranged from seven to 38 years. The average latent period was 15 years.

Symptoms

All persons complained of dyspnea and cough. Dyspnea was the major complaint and tended to progress gradually over the years. Cough and sputum production were mentioned by most but were less bothersome than the shortness of breath. Six persons complained of 'hemoptysis'. Specific details are not available in all cases, and thus this may represent anything from a once in a lifetime streak of blood, a major bleed or black sputum. Hemoptysis is not a commonly reported symptom of pneumoconiosis in general, nor of the inert dust diseases in particular. None of those who had 'hemoptysis' had tuberculosis nor any other condition, aside from chronic bronchitis which may be associated with hemoptysis. Whether the 'hemoptysis' was the result of chronic bronchitis or nepheline syenite induced changes is impossible to say, though it would seem to be more likely to be due to bronchitis. Several persons noted the occurrence of more frequent respiratory infections following development of their disease.

Physical Findings

Ten persons were reported to have crepitations on ausculation. Clubbing was noted in two persons. In one case, this was associated with radiological changes consistent with pulmonary osteoarthropathy. One person had culture-proven tuberculosis.

Pulmonary Function Tests

The pulmonary function testing has been done by a variety of methods over the years, and many of the results are from tests done 20 years ago. For the early tests only forced vital capacity (FVC) and maximal voluntary ventilation (MVV) were available, whereas recently a complete assessment of lung volumes and flows as well as diffusing capacity and exercise testing has been done. Thus, the completeness of information varies widely from person to person.

The pulmonary function test results revealed a diversity of findings. Of the 11 persons for whom results are available, nine had obstructive changes, five had restrictive changes and five had abnormalities in their diffusing capacity. Three persons had both restrictive and diffusion defects. Thus, a variety of abnormalities may be found.

Radiological Changes

The x-ray findings were relatively characteristic. Initially, there is diffuse pinhead mottling of the lung fields which progresses eventually into areas of conglomeration.

Ten cases had both changes. Three others had one of the two characteristic changes, pinhead mottling or areas of conglomeration. The conglomeration develops generally after the diffuse changes and progresses gradually. The areas of conglomeration were varied: eight were in the lower zones only, one was in the upper zones only and two were in both zones. There was one person who developed the diffuse pinhead mottling with early suggestions of conglomeration which then progressed to diffuse honeycombing. One person had neither of the typical findings but had evidence of emphysema, which may have obscured the parenchymal abnormalities.

It is difficult to ascertain the latent period between the initial exposure in the old mill and the chest x-ray changes because approximately one-half had abnormalities on the earliest x-ray which was available for review. In these cases, the changes may have been present for several years.

Pathology

Pathological data were available for five persons. One of these is not included in this series of 14 persons, because no clinical history, pulmonary function tests or x-rays were available, and he did not receive compensation. He died of a bronchogenic carcinoma and had been exposed to nepheline syenite dust for 25 years of which six were spent in the old mill. The pathology reports consist of two biopsy and four postmortem studies.

Common findings included replacement of the lung parenchyma with tissue containing many macrophages and dense accumulations of dust particles. Dust was found both extracellularly in the alveoli and interstitium, and within macrophages. The macrophages were numerous and many contained pigment, some of which was birefringent. Analysis of the dust by x-ray diffraction revealed it to be similar to the dust to which the worker had been exposed in the mill. The dust particles tended to aggregate around small arteries, arterioles and bronchioles. There was often marked hyperplasia or thickening of the vessel walls. Occasionally, the infiltrate formed aggregates which

were like lymphoid nodules. Where the dust concentration was smaller, there were some reticulin fibres but minimal fibrosis. Where the dust was more concentrated, fibrosis in varying amounts was present. There were sparse infiltrations of plasma cells, lymphocytes and eosinophils in some areas. The hilar nodes often contained large quantities of dust but minimal fibrosis was noted.

One man developed a mass lesion in his right lower lobe. At thoracotomy an abscess and bronchiectasis were found distal to an obstruction of a segmental bronchus. This was caused by enlarged hilar lymph nodes filled with dust particles.

Outcome

Seven of the 14 persons have died. In one case, the cause of death was unknown; in two, the cause was respiratory; in three, it was cardiovascular and one person died from complications following surgery for Cushings syndrome.

Reports of Typical Cases

Case 1

Mr. B. was born in 1901 and worked on a farm and as a furnace repairman after leaving school. In 1936 he began to work in the nepheline quarry and in 1948, he became a crusher operator in the mill. He did this until 1963. He had eight years exposure in the old mill.

In 1961 he developed dyspnea on exertion and a productive cough. He was a non-smoker. He also complained of fatigue and weight loss. Physical examination was essentially normal. His symptoms have progressed gradually over the years, and he is presently dyspneic when walking on the level at his own pace.

Chest x-rays are shown in Figure I and show the typical findings and progression. Pulmonary function results reveal slight obstruction.

He represents a case with classical symptoms and radiological changes and minimal ventilatory abnormalities.

Case 7

Mr. P. was born in 1920 and worked on a farm until he was 21. Between 1941 and 1945 he worked in the nepheline quarry. He returned to farming between 1945 and 1950.

Figure 1A — Chest X-Ray 1966

Figure 2A — Chest X-Ray 1958

Figure 1B — Chest X-Ray 1977

Figure 2B — Chest X-Ray 1971

In 1950 he became a sweeper in the old mill and in 1953 he started to operate one of the separating machines there. He had a total of six years' exposure in the old mill.

In 1961 he developed dyspnea on exertion and a productive cough. He had occasional episodes of hemoptysis. He was a smoker. Physical examination revealed a few crepitations but was otherwise normal. His dyspnea has gradually increased so he can now only walk slowly on the level.

Chest x-rays are shown in Figure 2 and illustrate the typical findings. Pulmonary function results are shown in Table 2. They reveal moderate obstruction and a low diffusing capacity. The changes progressed over the years.

He represents a case with the typical symptoms and radiological changes and, in addition, some of the ventilatory abnormalities which may occur.

Discussion

The 14 cases reviewed experienced dust exposure in a nepheline syenite mill prior to 1956. There are no records of dust levels, but personal accounts suggest that the levels were very high. No cases have been found in persons who have worked only in the new mill. Workers reported that the dust was much less in the new mill. As there is now a potential 24 years of exposure in the new mill, there has been ample time for the development of the typical condition as the average latent period for symptoms in the old mill was 15 years. As no new cases have been reported, it seems that the only significant exposure occurred in the old mill. It may either have represented exposure to much higher levels of dust, or, less likely, exposure to a different size of dust which was in the respirable range. Though there is no evidence for it, one cannot exclude the possibility that there was some other material in the old mill which has led to this condition.

It seems reasonable to assume that the major difference in exposure between the old and new mill was the quantity rather than the quality of the dust. Furthermore, as high levels of exposure for relatively short periods of time (minimum four years) have led to disease, while exposure to lower levels over a longer period of time does not seem to have caused a problem, this suggests that the cause of injury is overloading of the dust clearance mechanisms rather than some specific injurious property of the dust itself.

This is supported by the fact that nepheline syenite is a non-fibrous silicate which contains no free silica, and thus would not be expected to be particularly fibrogenic.

The pathological findings are also consistent with the proposed cause of injury. There were accumulations of large amounts of dust particles, but there was generally minimal fibrotic reaction. This is consistent with the inhalation of large amounts of non-fibrogenic dust which overwhelms the pulmonary clearance mechanisms. This would be more likely to occur with exposures to very high levels of dust as were found in the old mill, rather than the lower dust levels in the new mill.

The findings in these cases are similar to those occurring in persons exposed to Fuller's earth[7] and Kaolin.[8,9] These are aluminum silicates. Most of the reports of pneumoconioses related to these substances occurred 20 or more years ago when conditions in the workplaces were apparently very dusty. The x-ray findings in kaolin workers are similar to those found in nepheline syenite exposed workers. The kaolin workers initially developed fine one- to two-millimetre discrete nodules throughout the lung fields. In some cases, these progressed to massive conglomerate shadows in the upper zones.[8] Pathological findings in both of these conditions include accumulations of macrophages filled with dust particles and generally little fibrosis.[7,8] These substances were thought to be inert dusts. The changes occurring in workers exposed to them were thought to be due to retention of the dust particles in the lungs, possibly because of an inadequate respiratory clearance mechanism in the presence of high concentrations of dust, rather than any fibrogenic quality of the dust itself. The nepheline syenite story appears to be similar.

No reliable information concerning the incidence or prevalence of this condition can be obtained from the available data. The number of persons employed in the old mill who received sufficient exposure, and the number of persons who actually developed changes consistent with the disease are not known. The case finding method was by review of the files for persons who had submitted a claim for compensation.

Thus anyone who had the disease but did not submit a claim has not been included. There is also the possibility that some people may have developed early radiological changes without functional impairment. There are available x-ray reports on other

workers who were employed in the old mill. Several of these noted the earliest changes of pinhead mottling, but they apparently did not progress to conglomeration. Thus many persons may have had some effects from their exposure, but did not develop symptoms sufficient to submit a claim. For these reasons, no reliable estimate of prevalence or incidence can be made.

One worker with six years' exposure in the old mill submitted a claim for compensation for the development of lung cancer. The claim was not allowed, as there is no known association between exposure to nepheline syenite and lung cancer. Obviously, no conclusions about the incidence of lung cancer in association with nepheline exposure can be made from this one case.

No other data are available concerning illness related to nepheline exposure. A communication from Norway[10] in 1975 stated that no cases of pneumoconiosis had been seen. This is not surprising as the dust levels in the Norwegian mill, which was built in the mid 1960s, should be much lower than those in the old Ontario mill.

Conclusion

Lung disease may result from exposure to high levels of nepheline dust. In such cases, large accumulations of dust and macrophages may be found in the alveoli, interstitium and lymph nodes. Little fibrosis is likely to be present. The changes are thought to result from a failure of the respiratory defences to clear excessive amounts of dust. Nepheline syenite pneumoconiosis presents with non-specific respiratory symptoms. A variety of pulmonary function abnormalities may be seen. The x-ray shows diffuse pinhead mottling which often progresses to conglomeration. All cases in Ontario, to date, have occurred in persons who worked in an old mill. It is not anticipated that any cases will develop in those persons who have only worked in the new mill, so the disease may soon become one of historical interest.

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Table 1

Case Number	1	2	3	4	5	6	7	8	9	10	11	12	13	14
Exposure														
Year commenced exposure	36	35	36	39	48	43	41	52	40	50	35	50	49	39
Total years' exposure	24	35	20	13	13	7	17	23	33	10	15	12	8	19
Total years – old mill	8	20	14	7	8	7	6	4	14	6	15	6	7	16
Latent Period	13	38	12	10	8	7	11	18	32	8	7	7	8	15
Smoker	–	X	X	X	X	?	X	–	X	X	?	?	?	?
Symptoms														
Dyspnea	X	X	X	X	X	X	X	X	X	X	X	X	X	X
Cough	X	X	X	X	X	X	X	X	X	X	X	X	X	X
Sputum	X	X	X	X	X	?	X	–	X	X	X	X	X	X
Hemotysis	–	–	–	X	–	?	X	–	X	X	–	X	X	–
Physical Findings														
Crepitations	X	–	X	–	X	X	X	–	–	X	X	X	X	X
Clubbing	–	–	–	–	–	X	–	–	–	–	X	–	–	–
X-ray														
Typical changes	X	X	X	X	X	X	X	X	X	–	X	X	X	X
Other	–	–	–	–	a	–	–	–	–	–	b	–	c	–
Lung Function														
Obstruction	X	X	X	X	X	?	X	X	X	–	–	X	?	?
Restriction	–	–	–	X	X	?	X	–	–	X	X	–	?	?
Diffusion defect	–	–	X	–	X	?	X	–	–	X	?	X	?	?
Death														
Age	–	–	70	–	70	?	–	–	–	–	54	55	35	76
Cause – respiratory	–	–	X	–	–	?	–	–	–	–	X	–	–	–
– cardiovascular	–	–	–	–	X	?	–	–	–	–	–	X	–	X
– other	–	–	–	–	–	?	–	–	–	–	–	–	X	–

Key
X present
– absent
? no information
a honeycombing
b pulmonary osteoarthropathy
c adenoma

Table 2
Pulmonary Function Results – Case 7

		1964	*1968*	*1971*
FVC (L)	Observed	3.6	3.45	2.7
	Per cent predicted	93 %	82 %	65 %
FEV_1 (L)	Observed	2.05	1.80	1.35
	Per cent FVC	57 %	52 %	50 %
MVV (L/Min)	Observed	92	54	36
	Per cent predicted	80 %	48 %	33 %
DC_0 (ml/mmHg/min)	Per cent predicted	80 %	58 %	50 %

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Nitroglycerin and Medical Surveillance

Summary

Nitroglycerin (glyceryl trinitrate) and ethylene glycol dinitrate (EGDN) are used in the manufacture of dynamite; nitroglycerin is also used in gunpowder and more recently as a solid fuel for rockets. Medical uses include treatment of angina pectoris and relief of esophageal spasm.

Nitroglycerin and EGDN act on the musculature of the arterioles and arteries producing peripheral vasodilation. Workers in the explosives industry and some pharmaceutical employees are exposed to the effects of these substances, acute and chronic, and in some instances experience withdrawal symptoms. Some authors have reported cases of nonatheromatous ischemic heart disease and sudden death in workers, presumed to be induced by chronic industrial exposure to nitroglycerin and related nitrates with subsequent withdrawal.

The literature is reviewed and recommendations are made for the establishment of a medical surveillance program.

Nitroglycerin was first synthesized in 1846 by Sobrero in Torino, Italy. Sobrero also investigated dynamite and served as a consultant to Alfred Nobel[1].

The substance came into general use with the development of the Nobel process (1860s) of absorbing nitroglycerin with diatomaceous earth. This classic type of dynamite turned out to be risky in the winter because nitroglycerin froze to crystals resulting in unexpected and severe accidents. Therefore, in the 1930s nitroglycol was introduced to lower the freezing point of dynamite, thereby giving technical and economic advantage.

Different proportions of nitroglycol and nitroglycerin have been used in dynamite manufacture in the past; presently a ratio of about 8:2 is used[2].

More recently nitroglycerin has been used as a solid fuel for rockets.[1]

The correct chemical name for nitroglycerin is glyceryl trinitrate. It belongs to the group of aliphatic nitrates which are nitric acid esters of monovalent and polyvalent aliphatic alcohols. The nitrate group has the structure $CONO_2$.

The nitric acid esters of the lower mono, di and trivalent alcohols are liquids and those of the quadrivalent alcohols and hexavalent alcohols are solids[3].

Nitroglycerin is one of these esters and is synthesized by adding glycerol to a cold mixture of concentrated nitric and sulfuric acids:

$$\begin{array}{l} CH_2OH \\ | \\ CHOH \\ | \\ CH_2OH \end{array} + 3\,HNO_3 \xrightarrow{H_2SO_4} \begin{array}{l} CH_2ONO_2 \\ | \\ CHONO_2 \\ | \\ CH_2ONO_2 \end{array} + 3H_2O$$

glyceryl trinitrate

The upper oily layer contains the product which is purified by washing with water and alkali to remove H_2SO_4, HNO_3, HNO_2 and nitrogen oxides[1].

Physical and Chemical Properties

Nitroglycerin (Table 1) is a liquid with a sweet burning taste. The molecular weight is 227. It crystallizes in two forms, a labile form which melts at 2.8°C and a stable form with a melting point of 13.5°C. Upon heating, nitroglycerin starts to decompose at 50°C, volatizes appreciably at 100°C, gives off yellow vapors at 135°C, and explodes at 218°C. Upon percussion, nitroglycerin explodes violently with the release of large volumes of gas as indicated by the equation:

$$4C_3H_5(ONO_2)_3 \rightarrow 12CO_2 + 10H_2O + 6N_2 + O_2$$

Nobel estimated that one volume of nitroglycerin releases approximately 10,000 volumes of gaseous explosion products.

Nitroglycerin is soluble in water to the extent of 1.25 mg/ml, much more soluble in alcohol, and completely miscible with organic solvents such as ether, ethyl acetate and benzene.

Although the vapour pressure of nitroglycerin is low, its saturation of air at 0.33 ppm is a current air pollution problem because the compound is absorbed so readily through the lipoidal surfaces of the lungs and skin[1]. Nitroglycerin can undergo two kinds of purely chemical reactions in vivo, hydrolysis and reduction.

Occupational Exposure

Occupations with potential exposure to nitroglycerin and ethylene glycol dinitrate include: chemical and explosives workers, pharmaceutical workers, dynamite makers, miners, missile technicians, munitions loaders, munitions workers, nitroglycerin workers, rocket fuel makers, shell fillers and smokeless-powder makers.[4]

Employees most at risk to exposure to nitroglycerin and ethylene glycol dinitrate are the men operating the nitrator, the nitroglycerin buggy operator who delivers measured quantities of the nitroglycerin-nitroglycol mixture to the mix house and workers in the mixing and cartridge-filling operations[5].

Dynamite Manufacture

The dynamite manufacturing process in explosive plants follows the standard pattern originally developed by Nobel. At present, a sensitizing component, a mixture of nitroglycol and nitroglycerin, is produced by nitrating a solution of ethylene glycol and glycerin in various proportions in the presence of the hydrating agent, sulfuric acid. (Up to 80 per cent nitroglycol

may be used. Originally, the sensitizing component was entirely nitroglycerin but the addition of ethylene glycol dinitrate (EGDN) resulted in a more stable product with a lower freezing point.)

Dynamite consists of various mixtures of the sensitizing oil and organic materials, termed "dope" in the trade. Dope consists of various combinations of oxidizing salts and fillers, such as wood pulp, bagasse, corn flour, corn starch and rice hulls. The oxidizing salts are ammonium nitrate and sodium nitrate. Other inorganic additions such as sodium chloride, ammonium chloride and sulfur may also be used for imparting especially desired physical properties[5].

Work is performed in widely separated buildings called "houses" surrounded by earth embankments. (See Table 2.) Each process house is limited to occupancy by two to five men. Nitroglycerin and ethylene glycol dinitrate are prepared in a closed process, piped by gravity into a holding tank, and removed by buggy to mix houses where a batch process results in a mixture of sensitizing oil and dope. This mixture is then placed in hods which in turn are carried by railroad to cartridge-filling houses where the cartridges are filled, either by hand or automation.

The cartridges are then taken to packaging houses where they are sealed with waterproofing material and thence moved to the magazine or loading platforms for shipping[5,6].

Uses

Glyceryl trinitrate is incorporated with ethylene dinitroglycol to manufacture dynamite, an industrial explosive. It is also used in gunpowder. More recently it has been used as a solid fuel for rockets. In the practice of medicine, it is used therapeutically for its vasodilating effect in the relief of angina pectoris.

Kinetics

Absorption It is known that nitroglycerin can be absorbed through the skin, by inhalation and also by ingestion. Skin absorption is probably the major route of absorption in the workplace.

Metabolism As early as 1883 and 1885, it was known that inorganic nitrate was a metabolite of nitroglycerin[1].

In 1965, experiments showed that nitroglycerin was transformed into 1,2 and 1,3-dinitroglycerin which appeared as urinary metabolites[7].

Clinical Effects

1) *Acute* Nitroglycerin and ethylene glycol dinitrate have an acute action on the musculature of the arterioles and arteries, producing peripheral vasodilation. The immediate effect is depression of both the systolic and diastolic blood pressure. Pains in the chest, abdomen and extremities and symptoms of general fatigue may appear as a result. Continued exposure to ethylene glycol dinitrate in low concentrations produces a progressive rise in the diastolic blood pressure from the previously depressed level without a comparable rise in the systolic blood pressure. If such exposure continues, the diastolic blood pressure may consistently be recorded over 90 mm Hg, with a net narrowing of the pulse pressure[5].

Nausea and vomiting with resulting fatigue and loss of weight, cyanosis, dermatitis and CNS effects with acute mania have been reported[6].

Workers engaged in the manufacture and use of dynamite and related explosives have been subject to acute toxic reactions with severe headache, tachycardia, hypotension, methemoglobinemia and alcohol intolerance. Pharmaceutical workers engaged in the manufacture of nitroglycerin have also reported the same symptoms[8].

Because relatively short production runs suffice to build up an adequate stock, pharmaceutical workers do not develop the same tolerance as munitions workers who handle the substance daily[9].

2) *Chronic* Habitual headache is a common complaint of explosives workers. It often occurs after entering a powder magazine and may increase in intensity while working in the chamber. Workers sometimes find it necessary to go into the open air to obtain relief. Lowered cerebrospinal pressure which is known to be an accompaniment of the fall in blood pressure (caused by the vasodilatory effect of nitroglycerin) may be an important factor in causing headaches[10].

Powder workers undergo a combination of acute and chronic exposure. Medical tests done at the beginning of the work week, after a weekend free from exposure, will record the gradual rise of the diastolic pressure over the work period. If the pulse pressure of a powder worker remains low under the immediate vasodilating effects of nitroglycerin during the work week, then the dynamics of the CVS have been adversely affected.

3) *Withdrawal* Withdrawal symptoms occurring in workers exposed to nitroglycerin have been reported[11]. After continuous exposure to nitroglycol or nitroglycol with nitroglycerin for a number of years, an interruption of exposure may result in chest pain. This symptom generally appears 24-72 hours after interruption of exposure. The pain is similar to angina pectoris; it often disappears shortly after re-exposure, but such an attack of pain may be followed by death (due to cardiac causes). Autopsy generally does not show significant signs of atherosclerotic changes in the heart. Qualitatively, nitroglycerin and nitroglycol appeared to induce the same withdrawal reactions in workers[12].

Ischemic Heart Disease

In 1972, Lange[13] reported the cases of nine patients who manifested nonatheromatous ischemic heart disease induced by chronic industrial exposure to nitroglycerin with subsequent withdrawal. The cases involved workers in a Wisconsin munitions facility who handled a 37 per cent nitroglycerin-cellulose mixture (to be fashioned into solid rocket propellant). Despite the use of rubber gloves, protective apron and ventilatory systems, there was still physical evidence of skin contact. (Some workers were completely intolerant to the acute exposure; the rest developed tolerance and frequently took measures to maintain the tolerance). The nine patients developed symptoms between 12 to 48 months after exposure. Coronary and digital arteriospasm was demonstrated in one patient studied during the withdrawal state, but the symptoms were readily reversed by nitroglycerin. The probable pathogenic mechanism is as follows:

Acute Response Acute but sustained skin and pulmonary exposure to organic nitrates induces vasodilatation, arterial hypotension, decreased venous return and tachycardia. Headache and orthostatic hypotension are common symptoms.

Chronic Response After days, weeks, or months, compensatory responses of vasoconstriction come into play as manifested by increase in diastolic blood pressure, decrease in pulse pressure and the relief of headaches or other acute symptoms. This may lead to fixed diastolic hypertension.

Withdrawal Withdrawal from the vasodilatory effects over a 48-72 hour period leaves the more persistent vasoconstrictor response unopposed and the resultant general arteriolar constriction becomes clinically manifest in the cutaneous and coronary arteries.

Intermittent chest pain at this time is non-exertional but relieved by nitroglycerin. Coronary insufficiency or acute myocardial infarction may result unless nitroglycerin is given. This period of vasoconstriction subsides after 96-120 hours.

Lange *et al* postulate that ischemic heart disease may result from a functional increase in smooth muscle tone of the coronary artery, and the induction of ischemia by chronic administration of potent, long-acting organic nitrate vasodilators and subsequent withdrawal or discontinuance[13].

Sudden Death

The literature contains a number of reports about sudden death, and a study by Hogstedt in Sweden suggested that there was a considerably excess mortality in ischemic heart disease among men with long-term exposure to explosives[14].

In Germany, three fatalities occurred in explosives workers after two to three days absence from work. The deaths were attributed to coronary disease but organic changes characteristic of coronary atherosclerosis were minimal or absent[5].

In 1963, Carmichael and Lieben[5] reported four cases of unexpected death in explosives workers (employed as dynamite packers and shovelers) in a Pennsylvania plant. This observed incidence of death in an estimated exposed population of 75 men (in a four-year period) was 15 times the expected, according to mortality statistics. Although the four deaths were attributed to coronary heart disease, the authors noted that this cause reflected an unusually high incidence of coronary fatalities in men in the age range 30-45 years. Each of the four people had been a dynamite packer.

Investigation of another small explosives manufacturing plant in Pennsylvania disclosed four similar sudden deaths in workers in a five-year period. All four victims were involved with dynamite packaging. Some had previously complained of chest pain. Their anginoid attacks tended to occur on the weekend or initial day of the work week.

In a third small explosives manufacturing plant several deaths believed to be cardiac in nature occurred in a 12-year period. A 32-year-old dynamite

packer died suddenly on a Monday morning. He had complained of occasional episodes of chest pain and dyspnea. Autopsy showed sclerotic narrowing of the coronary arteries with pinpoint lumina. (Such findings have been described in most autopsied cases of dynamite workers who died suddenly)[5].

Causal Theories for Sudden Death

1. The observed episodes of "acute myocardial infarction" (said clinically to be the cause of sudden unexpected deaths) were due to coronary artery vasospasm tending to occur after the vasodilating influences during work-week exposure conditions wore off.

2. The episodes of sudden death were due to peripheral vasodilatation after continued exposure to nitroglycol over a period of years resulting in cardiovascular collapse as a consequence of deficient return of blood to the right heart.

3. A third theory postulates that exposure to vasodilating substances over long periods of time may compromise the nutrient arterioles in the walls of the arteries, causing the deposition of hyaline connective tissue ultimately resulting in sclerosis of the coronaries indistinguishable at autopsy from arteriosclerosis from other causes.[5]

Early evaluation of health risks in nitroglycerin workers can be detected clinically by complaints of work-related headaches, an increase in the pulse rate of (average) eight beats/minute, a drop in systolic blood pressure of 10 mm Hg or more, and a drop in pulse pressure. (See Table 3.)

Medical Surveillance

1. Preplacement Examination Before commencing employment with nitroglycerin in an industrial or pharmaceutical setting, it is necessary to have a preplacement examination of the potential worker

a) to exclude from exposure those with a history or presence of the diseases or high-risk medical conditions listed below:

congenital heart disease

congestive heart failure

arteriosclerotic heart disease

endocarditis, myocarditis and other infections

valvular dysfunction

arrhythmia or ECG changes

myocardial infraction

arterial hypertension
alcoholism
migraine headaches
epilepsy
brittle diabetes
psychological problems
long-term drug use

b) to obtain a baseline blood pressure reading and resting pulse rate prior to employment.

II. Periodic Examination

Periodic examinations are necessary every two to three months for six months then six-monthly to detect any adverse effects such as:

1) symptoms of headache, chest pain, dizziness, weakness, etc.;

2) signs related to work exposure (e.g. decreased systolic blood pressure, decreased pulse pressure) which may preclude continued work with nitroglycerin;

3) appearance of any of the previously listed medical contraindications to employment which may necessitate transfer to another job.

Special investigations may include:

a) an ECG, coronary angiography or hospital investigation;

b) blood for estimation of nitrate esters by gas chromatography. Gas chromatography can detect blood levels as low as 0.2 nanograms nitroglycol/ml and 3 nanograms nitroglycerin/ml. Therefore, monitoring of exposure should be based on determination of nitrate esters in venous blood samples taken at random intervals of the work day. The presumed safe levels are:

   i) a nitroglycol blood level not exceeding 2 ng/ml to correspond to the suggested TLV of 1 mg/m^3 in the air

   ii) the maximum nitroglycerin blood concentration should not exceed 4 ng/ml to correspond to a TLV of 2 mg/m^3 in the air.[15]

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Special Studies and
Services Branch,
Ministry of Labour,
400 University Avenue,
8th Floor,
Toronto, Ont.
M7A 1T7

III. Action Levels

Employees should be removed from exposure if there is

a) an increase in pulse rate (average) of eight beats/minute;

b) a drop in systolic blood pressure of 10 mm Hg or more with a drop in pulse pressure, and/or

c) the physician deems that the symptoms and signs from examination of the worker indicate the need.

Table 1
Physical and Chemical Properties of NG and EGDN

Compound	*Nitroglycerin*	*Ethylene Glycol Dinitrate*
Formula	$C_3H_5O_3(NO_2)_3$	$C_2H_4O_2(NO_2)_2$
M.W.	227.09	152.06
Appearance	pale yellow liquid	yellowish liquid
Odour	slightly sweet	none
M.P.	13.3°C unstable form 2.0°C	−22.3°C
B.P.	256°C	197-200°C
Explosive point	270°C	114°C
S.G.	1.59	1.49
V.P. (20°C)	.00012 to .011 mm Hg	.038-.050 mm Hg
Solubility		
Water	slightly soluble	insoluble
Alcohol	soluble	soluble
Benzene	soluble	very soluble
Ether	very soluble	soluble
Acetone	very soluble	very soluble

(NIOSH)

Table 2
Dynamite Manufacturing Process Flow Chart[5]

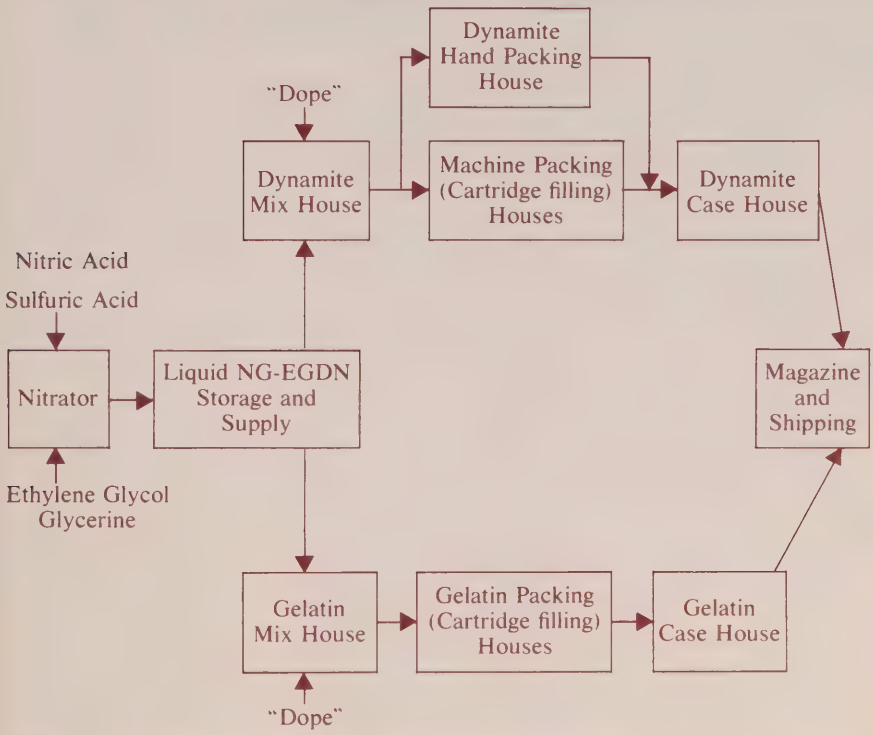

Table 3
Clinical Changes Found in Dynamite Plant Workers[6]

During Work	*After Work*
Unusual drop or rise in pulse rate	Unusually low or high pulse rate
Unusual drop in systolic blood pressure	
Unusual rise in diastolic blood pressure	
Unusual drop in pulse pressure	Unusually low pulse pressure. Complaint of headache

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A.H. Hall MD

Outpatient Treatment of Burns in Industry

Summary

An overview of outpatient treatment of burns is described. Certainly, there are areas which are controversial but if a routine is developed for decision making and treatment, the patient will benefit whether he is at home or in hospital.

Burns are among the most common injuries in industry. The majority of burns are minor, cause no disability and are easily handled. On the other extreme, the few severe burns are handled in burn units with the expert care they deserve. In between there is that burn which is certainly severe, painful and disabling but which does not reach the burn unit and is treated in the medical department. The literature in this area is scanty in comparison to burns treated in the burn unit, and certainly raises controversial issues. These involve such problems as to whether local antibiotics should be used, when and what systemic antibiotics should be used, whether blisters should be left intact, drained, or debrided, and whether open or closed methods should be used. This paper will deal with the evaluation of a burn, criteria for referral, and one example of a routine for outpatient care.

Etiology

1) *Thermal burns* from direct contact with open flame, hot metal, etc. are the most common.

2) *Chemical burns* are also common in industry and must be treated immediately and vigorously.

3) *Electrical burns* are ominous and should always be considered for referral to a plastic surgeon, as their depth and extent are difficult to ascertain when first seen.

4) *Radiation burns* must be considered as more x-ray and other radioactive sources are increasingly being used in industry.

Depth

It is somewhat difficult to ascertain the depth of a burn on first inspection but some estimation should be attempted when first seen, using the classification that you are comfortable with.

Classification of Burns

First degree burns Only the epithelium is involved and the injury presents as erythema. The burn is painful, always heals, and does not scar.

Second degree burns The epithelium and part of the dermis is destroyed. The dermal appendages, such as sweat glands and hair follicles are spared, allowing for re-epithelization. Superficial second-degree burns are painful, blistered, but usually heal without scarring. Deep second-degree burns may appear to be third-degree burns, they are not painful and heal in several weeks but with scarring. First- and second-degree burns can also be called partial-thickness burns.

Third-degree burns The entire thickness of the skin and its adnexae is destroyed. The burn is not painful, heals only from its margins and results in hypertrophic scarring.[1] If a third-degree burn is greater than 2-3 per cent of the body surfaces the patient should be referred to a plastic surgeon for possible grafting. This type of burn also is known as a full-thickness burn.

Criteria used for Outpatient Treatment

Adults with partial-thickness burns involving less than 15 per cent of the body surface can be considered for treatment on an outpatient basis.[2] However, as suspected, there are many exceptions:

a) Facial or neck burns should be admitted unless very superficial, especially if respiratory tract involvement is suspected.
b) Deep burns of the hand.
c) Burns of the feet or circumferential burns of the ankle.
d) Burns of the genital area and the perineum should be admitted because of obvious management problems.

Extent

A burn sheet should be adopted for each outpatient department. The "rule of nines" has stood the test of time and through the years has firmly established the practicality and validity of this system.[3] Each segment should be examined separately and the total arrived at by simple addition. Not only does it help you with the decision of whether the patient should be referred or treated as an outpatient, it also gives you a permanent record for comparison as the wound heals. It also helps with communication and, in hospital, certainly helps in the estimations for fluid replacement. (Refer to burn sheet Figure 1.)

Initial Treatment

A special area should be used in the outpatient department, especially away from areas where infected boils or other contaminated conditions are treated.

Patients with serious burns should be treated on a sterile sheet, giving special care to the airway, removing all the patient's clothing, applying sterile normal saline or cool water compresses to the area that is burned. Where possible, an IV normal saline or lactated Ringer's solution should be started in a peripheral vein. This also facilitates the use of analgesics, which are preferably given intravenously (3-5 mg of morphine) before transfer to the burn unit.

The treatment of less serious burns should be initiated with cool sterile normal saline compresses until the patient is comfortable. Intramuscular analgesics may be given as needed. After the initial pain is controlled the wound should be cleaned with a mild antiseptic soap or detergent (Savlon^{R}) and then irrigated with normal saline. Blisters should be left intact unless very large, in which case they should be carefully drained using sterile technique and the roof of the blister should be left as a biological dressing. If the wound is very dirty or grossly contaminated, thorough debridement should be carried out.

If the wound is facial, the open method is the preferred treatment. The wound should be cleaned twice daily with a mild antiseptic soap. The decision as to whether local antibiotics should be used must be made on clinical judgement. Most antibiotic ointments are frowned upon in burn units because of the emergence of resistant organisms, and because of the possibility of toxic effects due to absorption. This does not seem to be a problem in management in an Industrial medical department.

Most other wounds on an outpatient basis should be treated with the closed method, with the use of a sterile occlusive dressing. There is much opinion with little supportive data as to the value of topical agents under the dressing. It is probably safe to say: get to know how to use one or two topical agents efficiently and develop a routine for their use with which both you and your nursing staff are comfortable. The outer conforming dressing (Kling^{R}) should be bulky for a cushion effect as well as to absorb the drainage. Uniform compression can be maintained with elastic webbing dressing (Retalast^{R}, Surgifix^{R}).

All patients with burns run the risk of developing tetanus. A detailed history should be obtained as to

immunological status and the appropriate injection given. A guideline to follow: the patient who has had a full series of tetanus toxoid and who has had a recall injection within five years does not require immunization unless the wound is grossly contaminated. If the last booster was received between 5-10 years previously, the patient should have lcc of tetanus toxoid. Those whose immunological history is in doubt, or who have not had a full series should have a course of tetanus toxoid plus human tetanus immunoglobin.[4]

The patient should be given analgesics for home use and, if the extremities are involved, told to keep them elevated. The dressing should be changed periodically (48 hours) using sterile technique, watching closely for local and/or systemic infection. If the patient develops a low-grade fever the wound should be debrided, including blisters, a swab for culture and sensitivity taken and, depending on clinical judgement, systemic antibiotics which are effective for penicillin-resistant staphylococcus should be contemplated.

Once the wound is healed, a bland ointment such as lanolin will prevent itching, drying and blistering until the skin has "toughened up".

Special mention should be made of chemical burns. They should be thoroughly irrigated with cool tap water immediately. The chemical should be identified. If it is acid the irrigation can be stopped once the burning has settled. If it is alkaline, irrigation should continue by the clock for 30 minutes. If the burning remains severe and/or the wound has a soapy feel the irrigation should be continued for another 30 minutes. The nursing staff should be instructed to err on the side of over-irrigation. Many alkaline burns have converted from first-degree to third-degree overnight because of inadequate irrigation.

Hydrofluoric acid burns deserve special attention. It is safe to say that over-treatment is needed, as the burn is always more severe than it appears. A burn greater than 2½ per cent of body surface could lead to systemic poisoning by leaching of calcium.

The wound should be irrigated for at least 15-30 minutes with water. It should then be covered with a magnesium oxide dressing (20 per cent magnesium oxide). Finally, calcium gluconate can be injected (.5 cc per sq cm) around and under the wound when the burn is large or delayed. Injection is rarely necessary when the first aid treatment has been immediate and effective. If nails are involved they should be removed and the wound treated as above. The patient should

be referred for plastic surgical consultation after the initial treatment.[1]

Welders and burners in industry suffer two types of burns which are fairly unique.

1) Spark in the ear canal which, on first inspection, may not appear to cause too much damage. However, in 24 hours a significant number will show a perforated ear drum. Possibly all should be considered for an ENT consultation.

2) Sparks that burn through the pant leg and come to rest at the instep of the foot. The patient will have a severe burning sensation momentarily then the pain will disappear and he will continue to work. At the end of the day he will notice a painless third-degree burn. If he ignores the burn it will almost certainly become infected. Usually, however, such cases report to the outpatient department and some of these will require grafting to speed up the healing process.

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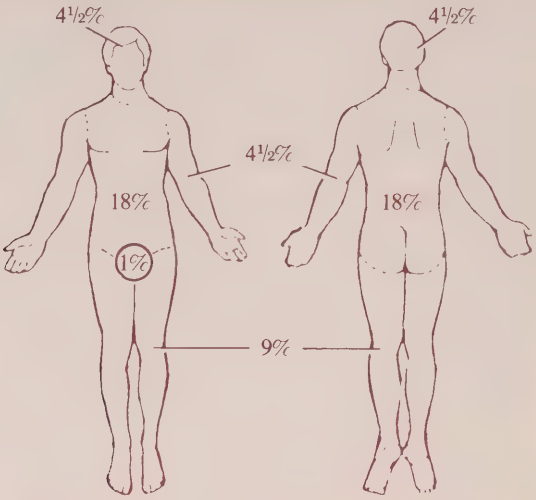
Acknowledgement

With thanks to Dr. E. S. Gibson, Director, Occupational and Environmental Health, for all his helpful suggestions.

*Adapted from Miller SF. Out Patient Management of Burns. *Am. Family Phys.* Nov. 1977; 119.

Figure 1



Weight (kg.) _____

Area	*Total %*
Head-Neck	
Anterior Trunk	
Posterior Trunk	
Right Arm	
Left Arm	
Right Leg	
Left Leg	
Total	

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Emergency Preparedness in The Ministry of Labour

Statement issued by the Emergency Planning Coordinator, Ministry of the Solicitor General, at the Emergency Preparedness for the Eighties Conference, November 16-19, 1980, Toronto.

The Occupational Health and Safety Division (OHSD) of the Ministry of Labour employs experienced staff with special knowledge of most aspects of environmental and occupational health and safety. These experts are prepared to respond to a wide variety of emergencies which might occur within the workplace, and to advise and assist other ministries in emergencies involving public health and safety.

The Mining Health and Safety Branch of the OHSD has overall responsibility for the mine rescue training program in Ontario. The training officers maintain a number of stations and substations equipped with breathing apparatus and fire fighting equipment as well as other emergency equipment and supplies to respond to a mine emergency.

The Construction Health and Safety Branch and the Industrial Health and Safety Branch are prepared to respond to most serious accidents and unusual situations. Medical advice, when required, is available from medical consultants in the Occupational Health Branch and the Special Studies and Services Branch.

The "Province of Ontario Contingency Plan for Spills of Oil or Other Hazardous Materials" indicates that in the event of spills or releases of toxic substances and in the event of release or escape of radioactive material the Ministry of Labour may be required to give advice to the Ministry of the Environment. For medical advice the ministry runs a 24-hour physicians' duty roster. Medical consultants from the Occupational Health Branch and the Special Studies and Services Branch, who are familiar with techniques for biological monitoring of workers and with the standards and guidelines for occupational and environmental limits to exposure to toxic substances, are available on 24-hour call through the Queen's Park emergency number.

The Ministry of Labour Special Studies and Services Branch also maintains a 24-hour emergency alert list specifically to handle situations involving the release or escape of radioactive material. These incidents include possible transportation accidents, industrial accidents including spills, and any other

unusual releases. Immediate advice is available over the telephone and, if required, survey and sampling teams may be sent to the site of the emergency to check for contamination and to advise on immediate actions.

The Radiation Protection Service and Radiation Protection Laboratory of the Special Studies and Services Branch play an integral role in the Province of Ontario Off-Site Nuclear Contingency Plan. Within its broad planning responsibilities the service prepares standing operating procedures and guides, for the Technical Advisory Group, the Survey Coordinating Group, the Radiation Protection Laboratory Staff and the Survey and Sampling Teams. Participating as members of the technical advisory group, assisted by experts from other government organizations, selected members of the Radiation Protection Service staff are the technical experts and advisers responsible for determining the severity and extent of the off-site releases and advising on remedial measures to minimize hazard to the public.

An essential part of the nuclear contingency organization is the Survey Coordinating Group (SCG) which is divided into three operational groups: a mobile field headquarters (located near the nuclear reactor site), a radio-chemical analysis laboratory (in Toronto) and a technical analysis section (with headquarters at the command post in Toronto).

The mobile field headquarters carries portable communication and monitoring equipment to outfit 10 survey and sampling teams manned by staff from the Ministries of Labour, Agriculture and Food, and Environment as well as Ontario Hydro personnel. The team leaders are radiation technicians from the Ministry of Labour staff. The field headquarters is also equipped with VHF radio and radio-telephone to permit transmission of data to the command post in Toronto. Samples collected from the field will be taken to the laboratory by prearranged air transport for detailed analysis.

The Radiation Protection Laboratory normally provides radio-chemical analyses of samples containing small amounts of radioactive material. In an emergency, when the samples to be analyzed require little preparation (e.g. water, milk and air filters), the laboratory can, for a limited time, analyze 16 samples per hour for gamma-ray emitters and 16 samples per hour for tritium. Output may be somewhat lower when samples require longer preparation or where radio-

chemistry and complex gamma spectra analysis are required. The results of the analyses are transmitted to the technical advisory group at the command post by telephone, VHF radio, radio-telephone or hard-copy transmitter.

The Analysis Section of the Survey Coordinating Group is prepared to calculate future concentrations of radionuclides (in air or water), future dose rates, doses and dose commitments on the basis of information available from time to time during a radiation emergency. This longer-term predictive role is to be distinguished from the role of the rest of the Survey Coordinating Group concerned with developing a comprehensive picture of the present (and near future) radiological situation. The predictive role requires calculation and modelling using, in addition to all the radiological raw data available, meteorological forecasts and lake current forecasts.

The Radiation Protection Service is also preparing to respond to nuclear contingency resulting from a release of radioactivity from a nuclear reactor in the United States. This response would be carried out in conjunction with federal agencies. Augmented survey and sampling programs are now being set up in regions of Ontario near U.S. nuclear reactor sites to provide data on background radiation levels and to facilitate effective response in the event of a contingency. The Ministry of Labour is assisted in these sampling programs by regional offices of the Ministry of the Environment.

News and Notes

In April and November of each year the Queen's University Occupational Health and Safety Resource Centre offers a training course in Industrial Audiometry and Hearing Conservation. This course has been designed to meet the Ministry of Labour's recommendations for appropriate training of audiometric technicians in industry. Students are recruited from among occupational health nurses and safety technicians, but those who supervise occupational health and safety programs would also benefit from the course. On successful completion of the course students should have a knowledge of the concepts of hearing conservation, an understanding of the physical measurement of sound and a practical ability to undertake competent audiometry. They will also be aware of the ethical and legal considerations surrounding audiometry and be familiar with record keeping techniques. For further information contact the Occupational Health & Safety Resource Centre, Queen's University, 25 Union Street, Kingston, Ontario. K7L 2N6.

Coming Events

March 26-27, 1981	Society of Occupational Medicine, Spring meeting, London, England.
April 7-10, 1981	BOHS Annual Conference 1981, University of Nottingham, England.
May 17-19, 1981	Third International Symposium on Hand-arm Vibration. Ottawa. Further information may be obtained from Mr. A.J. Brammer, Division of Physics, National Research Council, Ottawa. K1A 0R6. Tel: (613) 993-2840.
May 19-21, 1981	Canadian Association for Biological Safety. Conference on Biohazard Safety. Queen's University, Kingston, Ontario.
June 8-10, 1981	National Noise Control Conference, Raleigh, North Carolina.
August 16-19, 1981	International Symposium on Education and Training Policies in Occupational Safety and Health and Ergonomics. Organized by the Directorate of Labour Inspection of Norway in collaboration with the ILO and the WHO. Further information may be obtained from the International Labour Office, Occupational Safety and Health Branch, CH-1211 GENEVA 22.
September 25-30, 1981	20th International Congress of Occupational Health, Cairo, Egypt. For further information write to Professor Dr. Mahmoud Hassanein, Cairo University, 32 Falaki Street, Cairo.

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ISSN 0705-9388